	属する分野の分類(国際特許分類(IPC)) K67/027, A61K45/00, A61P25/16, C12N5/16, C	C12Q1/02, G01N33/15, G01N33/50 // C12	N15/12
調査を行ったは	行った分野 最小限資料(国際特許分類(I P C)) K67/027,A61K45/00,A61P25/16,C12N5/16,C	12Q1/02, G01N33/15, G01N33/50, C12N1	5/12
最小限資料以外	外の資料で調査を行った分野に含まれるもの		
国際調査で使り BIOSIS/WPI (D	用した電子データベース(データベースの名称 IALOG), PubMed, JSTPlus(JICST)	、調査に使用した用語)	
C. 関連する	ると認められる文献		
引用文献の カテゴリー*	. 引用文献名 及び一部の箇所が関連する	ときは、その関連する箇所の表示	関連する 請求の範囲の番号
A	Matsuoka Y. et al., Lack of nigral mice expressing human alpha-synuchydroxylase promoter. Neurobiol. Dis., 2001, 8(3), p. 535-9		1–13
A .	JP 2003-199460 A(東海林幹夫,他2名 全文(ファミリーなし)) 2003. 07. 15	1–13
× C欄の続き	とにも文献が列挙されている。		 紙を参照。
もの 「E」国際出際 以後に公 「L」優先権主 文献 (理 「O」口頭によ	のカテゴリー 車のある文献ではなく、一般的技術水準を示す 自目前の出願または特許であるが、国際出願日 会表されたもの E張に疑義を提起する文献又は他の文献の発行 は他の特別な理由を確立するために引用する 理由を付す) こる開示、使用、展示等に言及する文献 自目前で、かつ優先権の主張の基礎となる出願	の日の後に公表された文献 「T」国際出願日又は優先日後に公表さ出願と矛盾するものではなく、発の理解のために引用するもの 「X」特に関連のある文献であって、当の新規性又は進歩性がないと考え 「Y」特に関連のある文献であって、当上の文献との、当業者にとって自よって進歩性がないと考えられる 「&」同一パテントファミリー文献	語明の原理又は理論 語文献のみで発明 られるもの 語文献と他の1以 1明である組合せに
国際調査を完了	てした日 30.11.2004	国際調査報告の発送日 14.12.	.2004
日本国 理	0名称及びあて先 国特許庁 (ISA/JP) 『便番号100-8915 『千代田区霞が関三丁目4番3号	特許庁審査官(権限のある職員) 上條 発 電話番号 03-3581-1101	4B 3131 内線 3448

国際調査報告

C(続き).	関連すると認められる文献	
引用文献の カテゴリー*	引用文献名 及び一部の箇所が関連するときは、その関連する箇所の表示	関連する 請求の範囲の番号
. A	WO 01/60794 A2 (THE REGENTS OF THE UNIVERSITY OF CALIFORNIA) 2001.08.23, 特に, Example 7-9参照 & US 2004/0128706 A1	1-13
A	US 2002/0111321 A1(Ronald K.,他4名)2002.08.15 特に,Example 3参照 & WO 02/63951 A2 & EP 1418806 A2 & US 2004/0205833 A1	1-13
. A	WO 98/59050 A1(THE GOVERMENT OF THE UNITED STATES OF AMERICA represented by THE SECRETARY, DEPERTMENT OF HEALTH AND HUMAN SERVICES) 1998. 12. 30 特に,請求項63参照(ファミリーなし)	1-13
A	van der Putten H. et al., Neuropathology in mice expressing human alpha-synuclein. J. Neurosci., 2000, 20(16), p. 6021-9	1-13
A	Masliah E. et al., Dopaminergic loss and inclusion body formation in alpha-synuclein mice: implications for neurodegenerative disorders. Science, 2000, 287 (5456), p. 1265-9	i-13·
A	Richfield EK. et al., Behavioral and neurochemical effects of wild-type and mutated human alpha-synuclein in transgenic mice. Exp. Neurol., 2002, 175(1), p. 35-48	1-13
A	Kirik D. et al., Nigrostriatal alpha-synucleinopathy induced by viral vector-mediated overexpression of human alpha-synuclein: a new primate model of Parkinson's disease. Proc. Natl. Acad. Sci. U. S. A., 2003 Mar, 100(5), p. 2884-9	1-13
<u>A</u> .	Lo Bianco C. et al., alpha-Synucleinopathy and selective dopaminergic neuron loss in a rat lentiviral-based model of Parkinson's disease. Proc. Natl. Acad. Sci. U. S. A., 2002, 99(16), p. 10813-8	1–13
A .	Zhou W. et al., Overexpression of human alpha-synuclein causes dopamine neuron death in primary human mesencephalic culture. Brain Res., 2002, 926(1-2), p. 42-50	1-13
. A ,	Kanda S. et al., Enhanced vulnerability to oxidative stress by alpha-synuclein mutations and C-terminal truncation. Neuroscience, 2000, 97(2), p. 279-84	1–13

		
C (続き).	関連すると認められる文献	· · · · · · · · · · · · · · · · · · ·
引用文献の カテゴリー*	引用文献名 及び一部の箇所が関連するときは、その関連する箇所の表示	関連する 請求の範囲の番号
Α .	Park SM. et al., Distinct roles of the N-terminal-binding domain and the C-terminal-solubilizing domain of alpha-synuclein, a molecular chaperone. J. Biol. Chem., 2002, 277 (32), p. 28512-20	1-13
A	Kim TD. et al., Structural and functional implications of C-terminal regions of alpha-synuclein. Biochemistry, 2002, 41(46), p. 13782-90	1-13
PX	Ishi A. et al., Generation and analysis of transgenic mice that express human alpha-synuclein in dopamine neurons. 神経化学, 2004 Aug 10, 43(2, 3), p. 369, 0G1-06	1-13
T	Fernagut PO. et al., Alpha-synuclein and transgenic mouse models. Neurobiol. Dis., 2004 Nov, 17(2), p. 123-30	1-13
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第Ⅱ欄 請求の範囲の一部の調査ができないときの意見(第1ページの2の続き)
法第8条第3項 (PCT17条(2)(a)) の規定により、この国際調査報告は次の理由により請求の範囲の一部について作成しなかった。
1. □ 請求の範囲 は、この国際調査機関が調査をすることを要しない対象に係るものである。 つまり、
2. 図 請求の範囲 14-15 は、有意義な国際調査をすることができる程度まで所定の要件を満たしていない国際出願の部分に係るものである。つまり、特別ページ参照。
3. □ 請求の範囲は、従属請求の範囲であってPCT規則6.4(a)の第2文及び第3文の規定に 従って記載されていない。
第Ⅲ欄 発明の単一性が欠如しているときの意見(第1ページの3の続き)
次に述べるようにこの国際出願に二以上の発明があるとこの国際調査機関は認めた。
1. <u> </u>
2. 追加調査手数料を要求するまでもなく、すべての調査可能な請求の範囲について調査することができたので、追加調査手数料の納付を求めなかった。
3. 出願人が必要な追加調査手数料を一部のみしか期間内に納付しなかったので、この国際調査報告は、手数料の納付のあった次の請求の範囲のみについて作成した。
4. □ 出願人が必要な追加調査手数料を期間内に納付しなかったので、この国際調査報告は、請求の範囲の最初に記載されている発明に係る次の請求の範囲について作成した。
追加調査手数料の異議の申立てに関する注意
□ 追加調査手数料の納付と共に出願人から異議申立てがあった。 □ 22世間本工程は 0.444 と 世間 1.45 日間 1.45 日
□ 追加調査手数料の納付と共に出願人から異議申立てがなかった。

〈調査の対象について〉

請求の範囲14-15に係る「請求項12又は13に記載のスクリーニング方法により得られる物質」は、出願時の技術常識を勘案してもそのような性質を有する化合物の範囲を特定できないから、請求の範囲14-15は、PCT6条における明確性の要件を欠いている。

したがって、請求の範囲14-15に係る発明について有意義な調査をすることができない。

International application No.

PCT/JP2004/016373

A.	CLASSIFICA	TION OF SUBJECT	MATTER			
	Int.Cl7	A01K67/027,	A61K45/00,	A61P25/16,	C12N5/16,	C12Q1/02
		G01N33/15,	G01N33/50//	C12N15/12		

According to International Patent Classification (IPC) or to both national classification and IPC

B. FIELDS SEARCHED

Minimum documentation searched (classification system followed by classification symbols)
Int.Cl⁷ A01K67/027, A61K45/00, A61P25/16, C12N5/16, C12Q1/02,
G01N33/15, G01N33/50, C12N15/12

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched

Electronic data base consulted during the international search (name of data base and, where practicable, search terms used)
BIOSIS/WPI (DIALOG), PubMed, JSTPlus (JICST)

C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category*	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No:
A	Matsuoka Y. et al., Lack of nigral pathology in transgenic mice expressing human alpha-synuclein driven by the tyrosine hydroxylase promoter. Neurobiol.Dis., 2001, 8(3), p.535-9	. 1–13
A	JP 2003-199460 A (Mikio SHOJI et al.), 15 July, 2003 (15.07.03), Full text (Family: none)	1-13
A	WO 01/60794 A2 (THE REGENTS OF THE UNIVERSITY OF CALIFORNIA), 23 August, 2001 (23.08.01), Particularly, examples 7 to 9 & US 2004/0128706 A1	1-13

Further documents are listed in the continuation of Box C.	See patent family annex.		
Special categories of cited documents: document defining the general state of the art which is not considered to be of particular relevance	later document published after the international filing date or priority date and not in conflict with the application but cited to understand the principle or theory underlying the invention		
"E" earlier application or patent but published on or after the international filing date	"X" document of particular relevance; the claimed invention cannot be considered novel or cannot be considered to involve an inventive step when the document is taken alone		
"L" document which may throw doubts on priority claim(s) or which is cited to establish the publication date of another citation or other special reason (as specified)	"Y" document of particular relevance; the claimed invention cannot be considered to involve an inventive step when the document is		
"O" document referring to an oral disclosure, use, exhibition or other means "P" document published prior to the international filing date but later than	combined with one or more other such documents, such combination being obvious to a person skilled in the art		
the priority date claimed	"&" document member of the same patent family		
Date of the actual completion of the international search	Date of mailing of the international search report		
30 November, 2004 (30.11.04)	14 December, 2004 (14.12.04)		
Name and mailing address of the ISA/	Authorized officer		
Japanese Patent Office			
Facsimile No. Telephone No.			
Enra DCT/ICA/110 (conned choot) (Tonisors 2004)	Form DCYT/ISA/710 (conned cheat) (Tonuery 2004)		

International application No.
PCT/JP2004/016373

	Relevant to claim N
US 2002/0111321 A1 (Ronald K. et al.), 15 August, 2002 (15.08.02), Particularly, example 3 & WO 02/63951 A2 & EP 1418806 A2 & US 2004/0205833 A1	1-13
WO 98/59050 A1 (THE GOVERNMENT OF THE UNITED STATES OF AMERICA represented by THE SECRETARY, DEPERTMENT OF HEALTH AND HUMAN SERVICES), 30 December, 1998 (30.12.98), Particularly, Claim 63 (Family: none)	1-13
van der Putten H. et al., Neuropathology in mice expressing human alpha-synclein. J.Neurosci., 2000, 20(16), p.6021-9	1-13
Masliah E. et al., Dopaminergic loss and inclusion body formation in alpha-synuclein mice: implications for neurodegenerative disorders. Science, 2000, 287(5456), p.1265-9	1-13
Richfield EK. et al., Behavioral and neurochemical effects of wild-type and mutated human alpha-synuclein in transgenic mice. Exp.Neurol., 2002, 175(1), pages 35 to 48	1-13
Kirik D. et al., Nigrostriatal alpha- syncleinopathy induced by viral vector- mediated overexpression of human alpha- synuclein: a new primate model of Parkinson's disease. Proc.Natl.Acad.Sci.U.S.A., 2003 March, 100(5), p.2884-9	1-13
Lo Bianco C. et al., alpha-Synucleinopathy and selective dopaminergic neuron loss in a rat lentiviral-based model of Parkinson's disease. Proc.Natl.Acad.Sci.U.S.A., 2002, 99(16), p.10813-8	1-13
Zhou W. et al., Overexpression of human alpha- synuclein causes dopamine neuron death in primary human mesencephalic culture. Brain Res., 2002, 926(1-2), pages 42 to 50	1-13
Kanda S. et al., Enhanced vulnerability to oxdative stress by alpha-synuclein mutations and C-terminal truncation. Neuroscience, 2000, 97(2), p.279-84	1-13
	Particularly, example 3 & WO 02/63951 A2 & EP 1418806 A2 & US 2004/0205833 A1 WO 98/59050 A1 (THE GOVERNMENT OF THE UNITED STATES OF AMMERICA represented by THE SECRETARY, DEPERTMENT OF HEALTH AND HUMAN SERVICES), 30 December, 1998 (30.12.98), Particularly, Claim 63 (Family: none) van der Putten H. et al., Neuropathology in mice expressing human alpha-synclein. J.Neurosci., 2000, 20(16), p.6021-9 Masliah E. et al., Dopaminergic loss and inclusion body formation in alpha-synuclein mice: implications for neurodegenerative disorders. Science, 2000, 287(5456), p.1265-9 Richfield EK. et al., Behavioral and neurochemical effects of wild-type and mutated human alpha-synuclein in transgenic mice. Exp.Neurol., 2002, 175(1), pages 35 to 48 Kirik D. et al., Nigrostriatal alpha-synucleincyathy induced by viral vector-mediated overexpression of human alpha-synuclein: a new primate model of Parkinson's disease. Proc.Natl.Acad.Sci.U.S.A., 2003 March, 100(5), p.2884-9 Lo Bianco C. et al., alpha-Synucleinopathy and selective dopaminergic neuron loss in a rat lentiviral-based model of Parkinson's disease. Proc.Natl.Acad.Sci.U.S.A., 2002, 99(16), p.10813-8 Zhou W. et al., Overexpression of human alpha-synuclein causes dopamine neuron death in primary human mesencephalic culture. Brain Res., 2002, 926(1-2), pages 42 to 50 Kanda S. et al., Enhanced vulnerability to oxdative stress by alpha-synuclein mutations and C-terminal truncation. Neuroscience, 2000,

International application No.
PCT/JP2004/016373

		PCT/JP2	004/016373
C (Continuation).	DOCUMENTS CONSIDERED TO BE RELEVANT		
Category*	Citation of document, with indication, where appropriate, of the relev	ant passages	Relevant to claim No.
A	Park SM. et al., Distinct roles of the N-terminal-binding domain and the C-termina solubilizing domain of alpha-synuclein, a molecular chaperone. J.Biol.Chem., 2002, 277(32), p.28512-20	al-	1-13
A	Kim TD. et al., Structural and functional implications of C-terminal regions of alg synuclein. Biochemistry, 2002, 41(46), p.13782-90		1-13
P,X	Ishi A. et al., Generation and analysis of transgenic mice that express human alphasynuclein in dopamine neurons. Bulletinof the Japanese Society for Neurochemistry, 10 August, 2004 (10.08.04), 43(2,3), p.369,0G1-06	-	1-13
T	Fernagut PO. et al., Alpha-synuclein and transgenic mouse models. Neurobiol.Dis., 2004 November, 17(2), p.123-30		1-13
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International application No.
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Box No. II	Observations where certain claims were found unsearchable (Continuation of item 2 of first sheet)
1. Claims	l search report has not been established in respect of certain claims under Article 17(2)(a) for the following reasons: Nos.: e they relate to subject matter not required to be searched by this Authority, namely:
because extent	Nos. 14-15: they relate to parts of the international application that do not comply with the prescribed requirements to such an that no meaningful international search can be carried out, specifically: ra sheet.
3. Claims because	Nos.: e they are dependent claims and are not drafted in accordance with the second and third sentences of Rule 6.4(a).
Box No. III	Observations where unity of invention is lacking (Continuation of item 3 of first sheet)
1. As all reclaims.	equired additional search fees were timely paid by the applicant, this international search report covers all searchable
any add	carchable claims could be searched without effort justifying an additional fee, this Authority did not invite payment of itional fee. some of the required additional search fees were timely paid by the applicant, this international search report covers use claims for which fees were paid, specifically claims Nos.:
4. No requirestricted	sired additional search fees were timely paid by the applicant. Consequently, this international search report is ad to the invention first mentioned in the claims; it is covered by claims Nos.:
Remark on Prot	The additional search fees were accompanied by the applicant's protest. No protest accompanied the payment of additional search fees.

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Continuation of Box No.II-2 of continuation of first sheet(2)

(Subject of search)

With respect to the "substance obtained by the screening method of claim 12 or 13" claimed in claims 14-15, the scope of compounds with such characteristic cannot be specified even if technical common knowledge at the filing of the application is taken into account. Consequently, claims 14-15 fail to satisfy the requirement of clarity prescribed in PCT Article 6.

Therefore, no meaningful search can be conducted on the invention of claims 14-15.